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Chapter 1

Importance of Allelopathy as Peudo-Mixotrophy for the Dynamics and Diversity of Phytoplankton

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1. Introduction

Phytoplankton are responsible for oceanic primary production and oxygen generation; and essential for regulating the global carbon cycle [1]. The dynamics and diversity of phytoplankton are constrained by several top-down and bottom-up effects. Complexities further arise from inter-species interactions within phytoplankton communities. Resources available for the growth of phytoplankton (e.g., light and dissolved nutrients) are often limited. But, despite the presence of limited variety of resources, phytoplankton are capable of maintaining an extreme level of species diversity [1–3]. This diversity is paradoxical to the theory of competitive exclusion [3], which suggests that in the steady state the number coexisting species cannot exceed the number of limiting resources [4, 5]. The mechanisms proposed to explain phytoplankton diversity include environmental fluctuations, periodic fluctuations, spatial heterogeneity, deterministic chaos, life cycles, grazing, and chemical interactions (detailed in [6]). But, when the top-down effects and external factors are negligible, it is difficult to explain the 'building block' of the extreme diversity of phytoplankton, i.e., the stable coexistence of two phytoplankton on a single limiting resource.

There is a growing body of evidence, both theoretical and experimental, suggesting that allelopathic interactions among phytoplankton species have a major role in shaping phytoplankton-zooplankton dynamics and regulating phytoplankton diversity [6–23]. Some of these studies [20, 21] suggested that 'toxin-allelopathy' can prevent competitive exclusion in Lotkta-Volterra interactions. Further, the allelopathic effect can potentially mediate resource competition in a chemostat. Focusing on simple resource-competition models, Roy [22] proposed that two phytoplankton can stably coexist on a single resource in a homogeneous media without any external factors when allelopathy acts as 'pseudo-mixotrophy'. This chapter elucidates how this mechanism ('if you cannot beat them or eat them, just kill them by chemical weapons' [22]) determines the outcome of resource

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competition between two phytoplankton, and how it potentially contributes to maintaining phytoplankton diversity in natural waters.

2. Mixotrophy and allelopathy

Mixotrophy is known to influence species interactions within a food web [24]. Mixotrophic algae that can combine phototrophy and phagotrophy are an important component of phytoplankton communities (e.g., [25]). Mixotrophy can be an effective strategy for securing essential carbon required for the survival of algae in adverse conditions, such as, low radiation, unfavourable temperature, salinity or pH [26, 27]. Studies further suggested that certain algae (e.g., Prymnesium) can simultaneously be toxin producer and mixotrophic to 'kill and eat' [28]. However, not many species are known to follow this dual strategy that combines allelopathy and mixotrophy. But, several species are known to be allelopathic as they produce toxic or allelopathic chemicals (e.g., [13]). Studies suggested that the dynamics of phytoplankton with competitors and grazers are modulated by the presence of toxic species (e.g., [21, 29–31]). Allelopathy of toxin producers affects the growth and competitive ability of sensitive species. Allelopathy alone can potentially overturn the outcome of interspecific competition by providing 'additional' competitive and growth advantages to the allelopathic species [20, 22]. Roy [22] proposed that theoretically the effect of allelopathy can be viewed as pseudo-mixotrophy for the survival or coexistence of phytoplankton in nutrient competition. In the rest of the chapter, this mechanism will be discussed.

3. Allelopathy mediating competition for a single nutrient

To demonstrate the allelopathic effect on nutrient competition, a standard resource-competition model (presented in Table 1) is considered, which is a generalised version of the model analysed by [22].

Eq. (1): Nutrient	$\frac{dN}{dt}$	=	$\overbrace{d\,(N_0 - N)}^{\text{net nutrient input}} - \overbrace{\frac{1}{\eta_1} f_1(N)\,P_1}^{\text{uptake by } P_1} - \overbrace{\frac{1}{\eta_2} f_2(N)\,P_2}^{\text{uptake by } P_2} + \overbrace{\eta\,\alpha_1\,(m_1 P_1 + \phi(P_1, P_2)\,P_1)}^{\text{recycling from } P_1} + \overbrace{\eta\,\alpha_2\,(m_2 P_2)}^{\text{recycling from } P_2}$
Eq. (2): Non-allelopathic species	$\frac{dP_1}{dt}$	=	$\overbrace{f_1(N)\,P_1}^{\text{growth}} - \overbrace{m_1 P_1}^{\text{loss}} - \overbrace{\phi(P_1, P_2)\,P_1}^{\text{loss by allelopathy}}$
Eq. (3): Allelopathic species	$\frac{dP_2}{dt}$	=	$\overbrace{f_2(N)\,P_2}^{\text{growth}} - \overbrace{m_2 P_2}^{\text{loss}}$

Table 1. Representation of allelopathy in a nutrient-competition model of two phytoplankton

Parameter	Meaning	Unit	Functions/values from [22]
$f_1(N)$	Nutrient uptake function for species 1	day^{-1}	$\frac{\mu_1 N}{K_1+N}$
$f_1(N)$	Nutrient uptake function for species 2	day^{-1}	$\frac{\mu_2 N}{K_2+N}$
$\phi(P_1,P_2)$	Loss rate of species 1 due to allelopathy	day^{-1}	$\gamma P_1 P_2^2$
μ_1	Maximum growth rate of species 1 (P_1)	day^{-1}	1.0
μ_2	Maximum growth rate of species 2 (P_2)	day^{-1}	1.1
K_1	Half-saturation constant for species 1	gL^{-1}	0.6
K_2	Half-saturation constant for species 2	gL^{-1}	1.5
m_1	Per capita loss rate for species 1	day^{-1}	0.012
m_2	Per capita loss rate for species 2	day^{-1}	0.01
d	Dilution rate	day^{-1}	0.25
N_0	Input nutrient concentration	gL^{-1}	0.11
γ	Allelopathy parameter	$\text{cell}^{-3}\,\text{day}^{-1}$	0.02
α_1	Nutrient content per cell of species 1	$g\text{cell}^{-1}$	5×10^{-5}
α_2	Nutrient content per cell of species 2	$g\text{cell}^{-1}$	1×10^{-5}
η	Recycling efficiency	dimensionless	0.5
η_1	Yield coefficient of species 1	dimensionless	1.0
η_2	Yield coefficient of species 2	dimensionless	1.0

Table 2. Functions and parameters with their meanings used in the nutrient competition model with allelopathic effect. The quantities N, P_1 and P_2 are the concentrations of the nutrient, non-allelopathic species and allelopathic species, respectively.

The nutrient (with concentration N) uptakes by the non-allelopathic species (with concentration P_1) and allelopathic species (with concentration P_2) are described by the functions $f_1(N)$ and $f_2(N)$. In particular, these functions can take the standard Michaelis-Menten forms (Table 3). The parameters of the model are described in Table (2). Allelopathy of species 2 imposes a higher mortality to the non-allelopathic species, which can be described by an 'additional' mortality term in the form of a phenomenological function $\phi(P_1,P_2)$. This function may be a high-order interspecific product of P_1 and P_2 (see, Table 3) - a particular case of which was considered in [22]. In the absence of allelopathy, the model takes the form of a standard resource-competition model, which predicts the persistence of one of the two species depending on the lowest minimum nutrient requirements (i.e., depending on minimum R^* [5]). So, if $\phi(P_1,P_2)=0$, and if the non-allelopathic species has a lower minimum nutrient requirement, it will win over the allelopathic species in nutrient competition. However, if $\phi(P_1,P_2)\neq 0$, allelopathy provides advantage to species 2 by imposing a higher mortality to species 1.

3.1. Coexistence of two phytoplankton on single nutrient

As mentioned in the previous section, the loss rate of species 1 due to allelopathy of species 2 can be described by a high-order product of P_1 and P_2: $\phi(P_1,P_2)=\gamma P_1^{\beta_1} P_2^{\beta_2}$. A particular case was analysed in [22], where the exponents were taken as $\beta_1=1$ and $\beta_2=2$. For $\phi(P_1,P_2)=\gamma P_1^{\beta_1} P_2^{\beta_2}$, it can be derived (following the analysis of [22]) that there exist a critical value γ^c for the allelopathy parameter γ, such that, if $\gamma<\gamma^c$, no coexisting steady state is possible. However, if $\gamma>\gamma^c$, two alternative steady states are possible,

and depending on the initial conditions the system will settle to one of the two steady states (see, Fig. 1). Therefore, for $\gamma > \gamma^c$ one can find suitable initial concentrations of P_1 and P_2 for which stable coexistence two phytoplankton on a single nutrient is possible: Fig. 1-(b) shows that in this case the ratio of P_2 to P_1 is stabilised to a non-zero value.

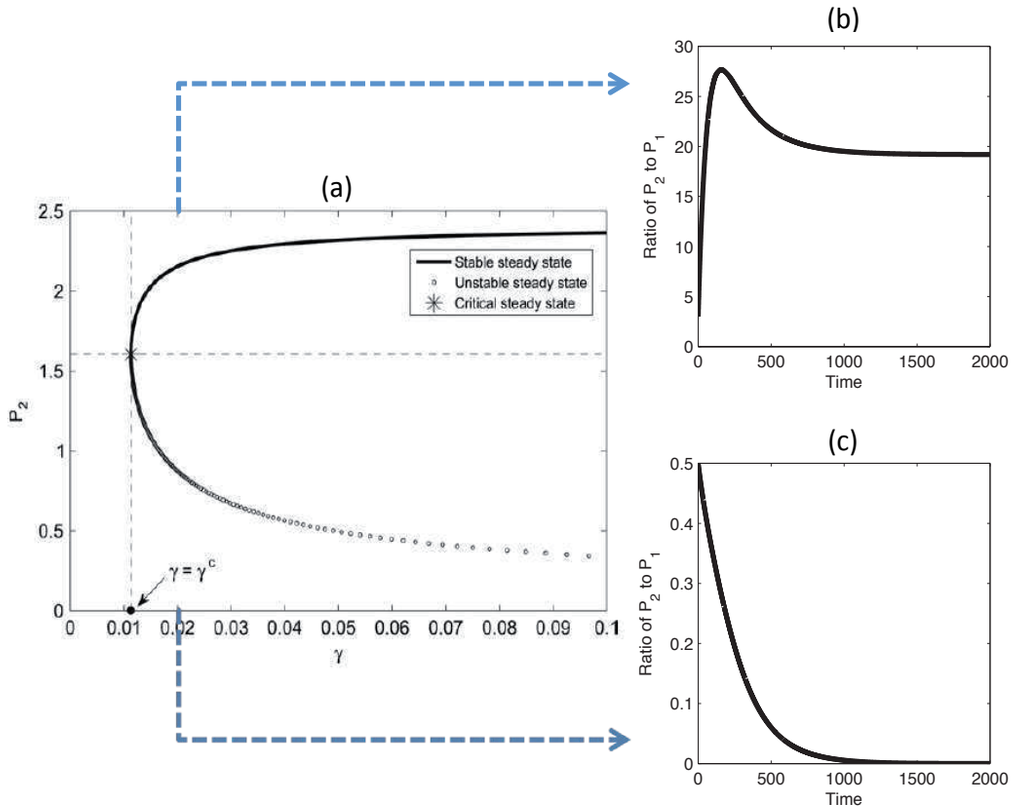

Figure 1. Dynamics two phytoplankton when allelopathy exceeds critical level. (a) Saddle-node bifurcation for the model system with γ as the bifurcation parameter. The figure was reproduced from [22] with permission from the publisher. (b) & (c) Ratio of P_2 to P_1 corresponding to the stable and unstable dynamics presented by (a), respectively. Condition for no recycling was used with other parameters and functions fixed at their default values/forms as in Table (2).

3.2. Critical conditions for coexistence

The critical level of allelopathy γ^c is a crucial quantity, which can be computed from the parameters of the model. Corresponding to γ^c, there exists an unique coexisting steady state $\left(N^*, P_1^c, P_2^c\right)$, where the magnitudes of P_1^c and P_1^c depend on the model parameters. Extending the analysis of [22] , the magnitudes of these quantities can be derived explicitly for all possible forms of the function $\phi(P_1, P_2)$ (Table 3). When $\gamma > 0$, the critical conditions for the existence of the unique steady state can alternatively be derived with respect to N_0 - the input nutrient concentration. The allelopathy parameter γ would depend on the inherent biological properties of the allelopathic species, and hence its magnitude cannot normally be altered using experimental conditions. However, the parameter N_0 associated with the experimental conditions can very well be controlled. Rearranging the expressions of γ^c (Table 3), one can derive the corresponding threshold magnitudes of the input nutrient concentration, say, N_0^c, so that, for $N_0 > N_0^c$ alternative steady states are possible leading to the stable coexistence of two phytoplankton. The explicit expressions of N_0^c for different forms of $\phi(P_1, P_2)$ are

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$\phi(P_1,P_2)$	(P_1^c,P_2^c)	γ^c for a given N_0	N_0^c for a given γ
γP_2	Does not exist	-	-
$\gamma P_1 P_2$	$\left(\frac{c_3}{2c_1},\frac{c_3}{2c_2}\right)$	$\frac{4Ac_1c_2}{c_3^2}$	$N^*+\left(\frac{4Ac_1c_2}{\gamma d^2}\right)^{\frac{1}{2}}$
$\gamma P_1 P_2^2$	$\left(\frac{c_3}{3c_1},\frac{2c_3}{3c_2}\right)$	$\frac{27Ac_1c_2^2}{4c_3^3}$	$N^*+\left(\frac{27Ac_1c_2^2}{4\gamma d^3}\right)^{\frac{1}{3}}$
$\gamma P_1^{\beta_1} P_2^{\beta_2}$	$\left(\frac{c_3\beta_1}{c_1(\beta_1+\beta_2)},\frac{c_3\beta_2}{c_2(\beta_1+\beta_2)}\right)$	$\frac{Ac_1^{\beta_1}c_2^{\beta_2}(\beta_1+\beta_2)^{(\beta_1+\beta_2)}}{c_3^{(\beta_1+\beta_2)}\beta_1^{\beta_1}\beta_2^{\beta_2}}$	$N^*+\left(\frac{Ac_1^{\beta_1}c_2^{\beta_2}(\beta_1+\beta_2)^{(\beta_1+\beta_2)}}{\gamma\beta_1^{\beta_1}\beta_2^{\beta_2}d^{(\beta_1+\beta_2)}}\right)^{\frac{1}{(\beta_1+\beta_2)}}$

Table 3. Parametric conditions for stable coexistence when allelopathy acts as pseudo-mixotrophy in nutrient competition models. The allelopathic effect is denoted by $\phi(P_1,P_2)$, the critical steady state by (N^*,P_1^c,P_2^c), the critical level of allelopathy by γ^c , and the threshold level of N_0 by N_0^c. The quantities c_1, c_2, c_3 and A used in the table are defined as: $c_1=\frac{1}{\eta_1}f_1(N^*)-\eta\alpha_1(m_1+A)$, $c_2=\frac{1}{\eta_2}f_2(N^*)-\eta\alpha_2 m_2$, $c_3=d(N_0-N^*)$, with $A=f_1(N^*)-m_1$, and N^* is given by $f_2(N^*)=m_2$. In particular, $f_1(N)=\frac{\mu_1 N}{K_1+N}$ and $f_2(N)=\frac{\mu_2 N}{K_2+N}$, $N^*=\frac{m_2K_2}{\mu_2-m_2}$.

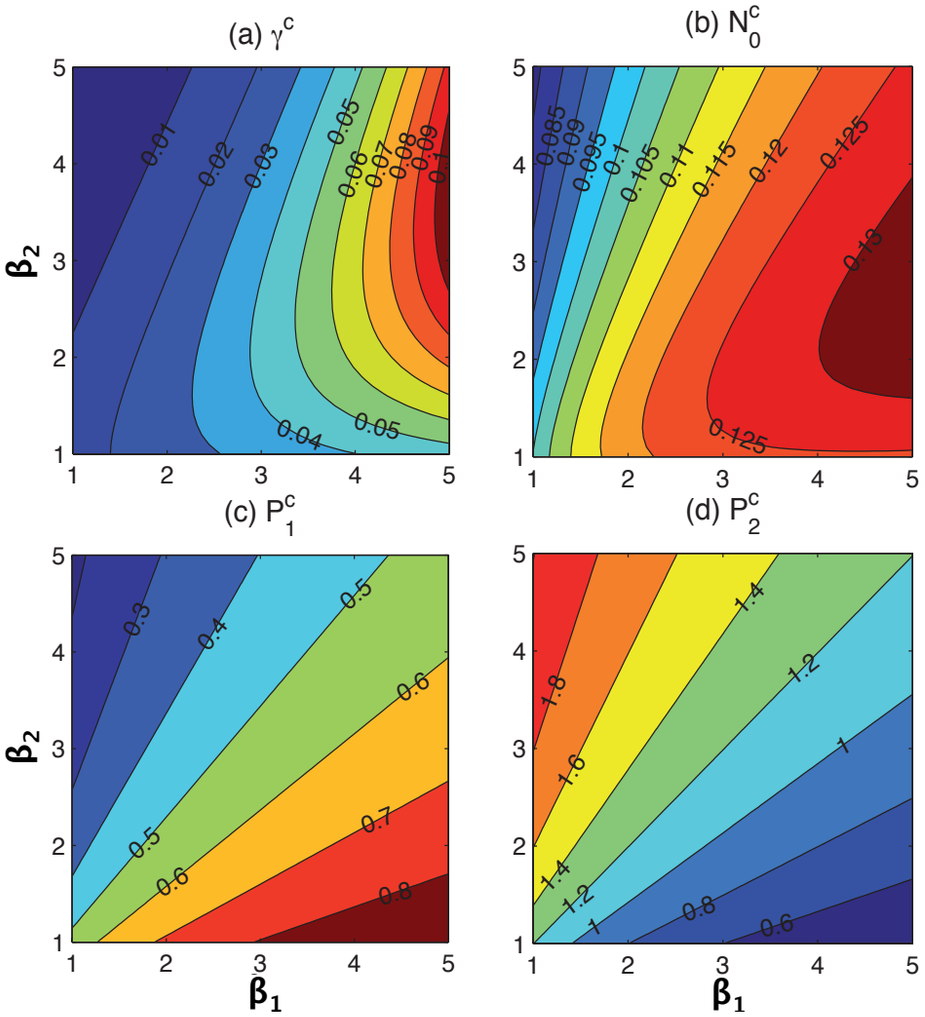

Figure 2. The magnitudes of γ^c, N_0^c, P_1^c and P_2^c are computed for a range of values of the exponents β_1 and β_2 corresponding to the function $\phi(P_1,P_2)=\gamma P_1^{\beta_1}P_2^{\beta_2}$. The parameters are fixed at their default values as in Table (2).

presented in Table (3). The results in Table (3) can be used to address how the critical values γ^c, N_0^c, P_1^c and P_2^c may change due to uncertainties in describing the allelopathic effect

by a phenomenological function. Considering the general form $\phi(P_1, P_2) = \gamma P_1^{\beta_1} P_2^{\beta_2}$, the magnitudes of γ^c, N_0^c, P_1^c and P_2^c are computed for a range of values of the exponents β_1 and β_2 (Fig. 2). If the model parameters are fixed, γ_c or N_0^c would be minimum when β_1 is the lowest and β_2 is the highest (Fig. 2-a, b). The unique steady states of P_1 and P_2 depend on both β_1 and β_1: for a given β_1, P_1^c decreases but P_2^c increases with β_2 (Fig. 2-c, d).

3.3. Allelopathy as pseudo-mixotrophy

The function of allelopathy in mediating the coexistence can be understood from the Figs. (3) & (4). Under nutrient-limiting conditions, allelopathy of the weaker competitor helps increase the availability of nutrient the by killing the stronger competitors: an illustration of this process based on the model of [22] is given in Fig. (3-a). In the simplest scenario,

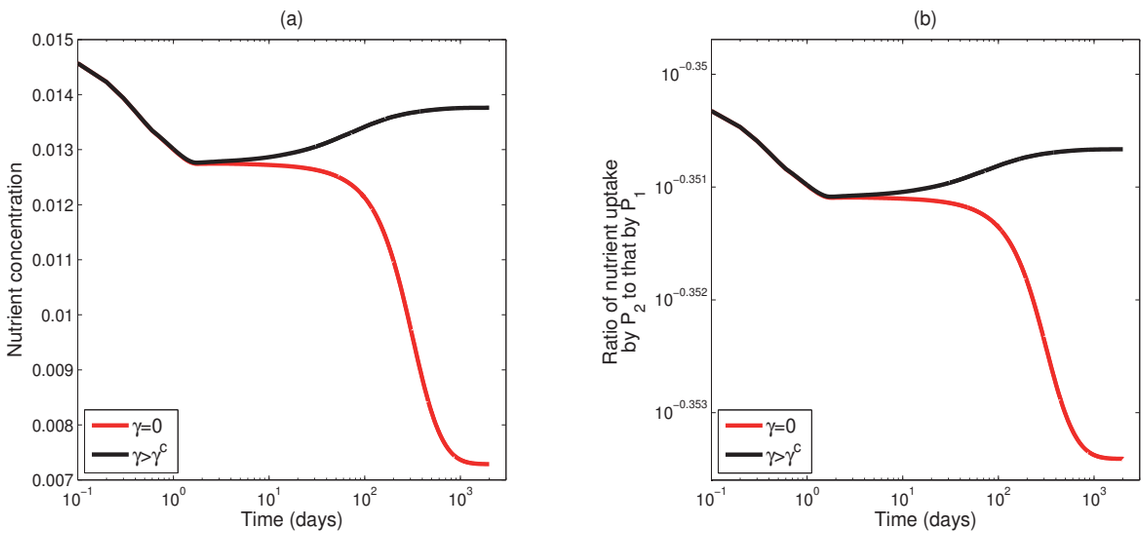

Figure 3. Allelopathy as pseudo-mixotrophy: (a) Enlargement of the available nutrient pool due to killing of competitors by allelopathy; (b) Increased ratio of per capita nutrient uptake by allelopathic species to that by non-allelopathic species. Red and black lines indicate conditions of no allelopathy and allelopathy beyond the critical level, respectively. Condition for no recycling was used with other parameters and functions fixed at their default values/forms as in Table (2).

when recycling of nutrient is 'turned off' in the model, and no killing by allelopathy takes place, the level of available nutrient decreases and stabilises to a low value where the non-allelopathic species alone survives eventually (Fig. 3-a, b). However, the extra (higher) mortality of species 1 (P_1) due to killing by allelopathy of species 2 (P_2) leads to elevation of the nutrient concentration (and further prevents it from decreasing gradually) (Fig. (3-a); the nutrient concentration eventually stabilises to a level where both species stably coexist (Fig. 3-a, b). The ratio of per-capita nutrient uptake by P_2 to that of P_1 decreases to a low value when killing by allelopathy does not take place (Fig. 3-b, Fig. 4-a); however, this ratio stabilises to a considerably higher value when allelopathy kills stronger competitors (Fig. 3-b, Fig. 4-b). When nutrient recycling is incorporated, killing by allelopathy increases the dead cells (Fig. 4-c,d), and the recycling process releases a portion of the nutrient quota of the dead competitors available for uptake (Fig. 4-d). The recycling process coupled with killing by allelopathy thus generates an extra amount of nutrient (Fig. 4-d) available for uptake by the species. Therefore, by imposing higher mortality to stronger competitor, allelopathy provides clear advantage to the weaker competitor. This mode of action of allelopathy

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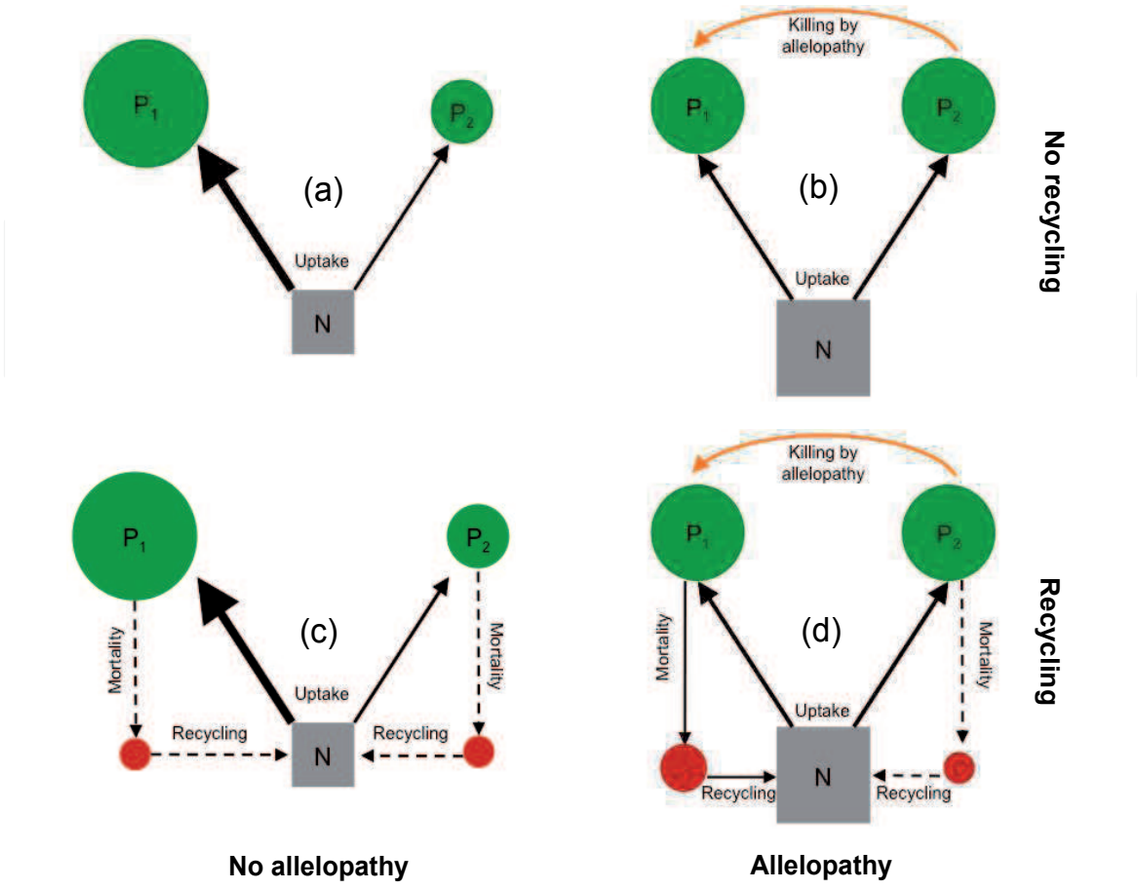

Figure 4. Schematic diagram showing the function of allelopathy as pseudo-mixotrophy. Competition between two phytoplankton (P_1, P_2) under a single nutrient (N) with or without the effect of allelopathy: (a) no nutrient recycling and no allelopathy; (b) no nutrient recycling, but killing by allelopathy; (c) nutrient recycling, but no allelopathy; and (d) nutrient recycling and killing by allelopathy. The thin and thick arrows indicate low and high values for nutrient uptake, respectively; dashed and continuous lines represent low and high level of recycling respectively; and the sizes of the circles and squares represent concentrations of the variables. The orange curved-arrows indicate when killing by allelopathy is incorporated. In (a) and (c), the weak competitor P_2 is excluded, whereas, P_1 survives. In (b) and (d), P_1 and P_2 stabilises with concentrations depending on the model parameters (which are not represented by the relative size of the circles).

that provides growth advantage to the allelopathic species, not through direct predation but through killing of the competitors (e.g., Fig. 4-b, Fig. 4-d), was termed as pseudo-mixotrophy [22]. In this process killing by allelopathy provides a positive feed-back by increasing of the growth limiting resource that reduces the competition pressure (e.g., Fig. 4). Clearly, this feedback loop provides crucial benefit to the growth rate of the allelopathic algae, and modulates the dynamics of the resource competition within a common trophic level.

4. Relevance to empirical and experimental studies

It is clear from the previous sections that allelopathy acting as pseudo-mixotrophy can theoretically stabilise nutrient competition of two phytoplankton on a single limiting nutrient. However, the applicability of this mechanism across natural phytoplankton is largely unexplored. An empirical or experimental evidence for pseudo-mixotrophy is

still in demand. But, recent studies have shown promise that the role of allelopathy in maintaining biodiversity of natural phytopalnkton may be explored further. For example, chemical warfare has been shown to increases bio-diversity in microbial realm [32]; and [20] showed that allelopathy may be responsible for co-existence of the competing phytoplankton in the Bay of Bengal. The question of how much diversity of phytoplankton can be supported though allelopathy alone was addressed by [23], who derived a deterministic relationship between the abundance of the potential allelopathic species and the diversity of non-allelopathic phytoplankton (see, Fig. 5). The abundance-diversity relationship in Fig.

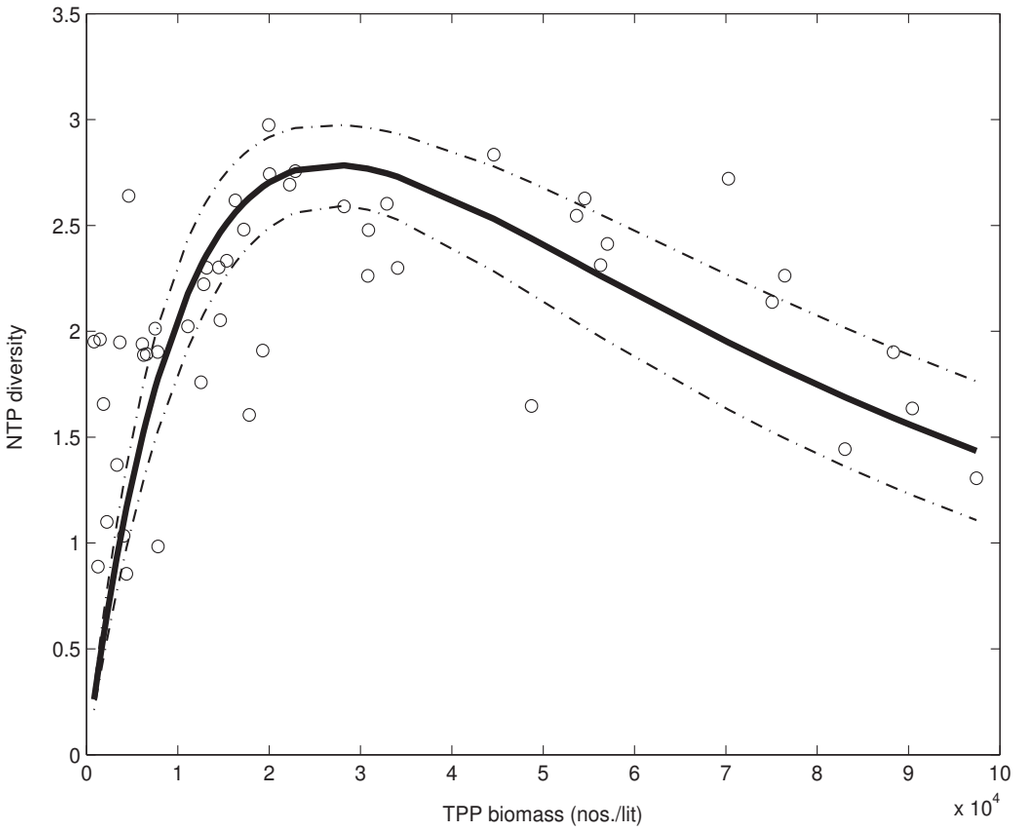

Figure 5. Deterministic relationship between the abundance of toxin-producing phytoplankton (TPP) and the diversity of non-toxic phytoplankton (NTP) in the Bay of Bengal. The figure is reproduced from [23] with permission from the publisher. The Shannon diversity of non-toxic species is plotted as a function of the abundance (nos./l) of toxin-producing phytoplankton defined by [23]. The solid line represents the fitted model of with the data presented in open circles. The dashed lines are the predicted model at 95% confidence level.

(5) shows a unimodal pathway through which the abundance of allelopathic phytoplankton regulates the diversity of non-allelopathic phytoplankton [23].

5. Concluding remarks

This chapter elucidates how phytoplankton allelopathy may function as pseudo-mixotrophy in determining the dynamics of nutrient-phytoplankton models, and how phytoplankton diversity is maintained in those systems. Firstly, the ecological conditions under which allelopathy functioning as pseudo-mixotrophy overturns the outcome of nutrient competition between two phytoplankton (e.g., [22]) is presented explicitly in terms of the model

parameters. Secondly, the difficulties in mechanistically describing the allelopathic effect of a phytoplankton on its competitors is addressed by considering a phenomenological function, and the ecological conditions for the coexistence of phytoplankton species and stability of competition dynamics are derived. Thirdly, the competition dynamics is explored under the assumptions of 'no nutrient recycling' and 'continuous nutrient recycling'; and the effects of changing initial nutrient pool in culture media is explored. Therefore, a comprehensive set of constraints is derived under which allelopathy acts as pseudo-mixotrophy in nutrient-phytoplankton models. Finally, the evidences of allelopathic effects in determining the diversity of phytoplankton in natural systems are presented. In particular, how the increasing abundance of allelopathic species may regulate the diversity of phytoplankton (e.g., [23]), is discussed. The mechanism presented here would be useful for better understanding of the biodiversity and function of marine ecosystems. Allelopathy functions as 'pseudo-mixotrophy' in nutrient-phytoplankton models, which are often the basis of marine biogeochemical and ecosystem models. This mechanism has not been explored in relation to ocean biogeochemical models, which are generally used to predict phytoplankton species composition, and estimate the scale of oceanic carbon sink. Given the complexities in representing phytoplankton functional types in global biogeochemical models (e.g., [33]), it would be useful to understand how allelopathy or pseudo-mixotrophy of a phytoplankton type may affect the dynamics of the other types. The ecological conditions derived will be useful for investigating the role of 'pseudo-mixotrophy' in marine ecosystem models. The current challenges in monitoring, controlling and managing harmful algal blooms (HAB) (e.g., [34]), and predicting their consequences in aquatic ecosystems require better understanding of the dynamics of toxic or allelopathic species. Recent studies have also reported other roles of phytoplankton allelochemicals, e.g., defence against predators [35], and 'casual parasitism' that helps supplying organic nutrient to the mixotrophic donors by lysis of prey [36, 37]. It will be worthwhile to further explore the mechanism presented here in relation to the succession of phytoplankton taxa that are known to form HABs. It is noteworthy that currently the mechanism has been explored using simple resource-competition models that can be tested in an experimental (chemostat) set up. Such an experiment will be helpful in formulating and parameterising resource-competition models including allelopathy, and for better understanding of the constraints of phytoplankton diversity.

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